UNIVERSITY OF MINNESOTA
SEP 11 1967
LIBRARY

VOLUME XIV April 1958 NUMBER 4

Clinical Proceedings

OF THE CHILDREN'S HOSPITAL

WASHINGTON, D. C.

A Review of Incidence, Complications, and Management of Eczema In Infants and Children. *Hyun-Wha Kim (Oh), M.D.* 77

Rectal Prolapse of an Ileocecal Intussusception. *Byron D. Roseman, M.D.* 89

Viruses as a Cause of Gastroenteritis. *Viola Mae Young, Ph.D.* 92

easier
antipyretic–analgesic
relief

you help her recover more easily
when you prescribe

Tempra

Acetaminophen, Mead Johnson

drops / syrup

Tempra is the first physician-controll
antipyretic–analgesic in two liquid dosa
forms–wild-cherry-flavored Drops a
mint-flavored Syrup. Both are readi
accepted, well tolerated and easy for t
mother to give, without forcing or fussin
Since Tempra is on Rx only, you ha
better control of medication and dosage.
parents have added confidence.

For detailed brochure on Tempra–you a
cordially invited to ask your Mead Johns
Representative or write us, Evansville 21, India

Mead Johnson
Symbol of service in medicine

MEAD JOHNSON & COMPANY, Suite 422 Eig Building, 8641 Colesville Road
Silver Spring, Maryland. JUniper 9-1222

CLINICAL PROCEEDINGS
OF THE CHILDREN'S HOSPITAL

2125 13th Street, N. W. Washington 9, D. C.

EDITOR FOR APRIL

GEORGE J. COHEN, M.D.

EDITOR-IN-CHIEF

ROBERT H. PARROTT, M.D.

EDITORIAL BOARD

FREDERIC G. BURKE, M.D.
JOSEPH M. LOPRESTI, M.D.
E. CLARENCE RICE, M.D.
SYDNEY ROSS, M.D.

MANAGING EDITORS

J. WILLIAM OBERMAN, M.D.
GEORGE J. COHEN, M.D.

ASSOCIATE EDITORS

JOHN BAYLY, M.D.
STANLEY L. BLUMENTHAL, M.D.
WINSTON E. COCHRAN, M.D.
GORDON W. DAISLEY, JR., M.D.
MAX FISCHER, M.D.
MILTON S. GLATT, M.D.
GRACE H. GUIN, M.D.
JOHN O. NESTOR, M.D.
MARSHALL M. PARKS, M.D.
GEORGE WILLIAM WARE, M.D.
CHARLES R. WEBB, M.D.
RALPH D. WHITLEY, M.D.

GENERAL MANAGER

THELMA WALLER

THE RESIDENT STAFF: AUGUSTO ALVARADO, M.D., SALVATORE BATTIATA, M.D., CATHERINE P. CHESTER, M.D., VICTOR S. CIPOLLA, M.D., MITHAT CORUH, M.D., ENRICO DAVOLI, M.D., DONALD W. DELANEY, M.D., GLORIA D. ENG, M.D., JOSEPH CHARLES EVERS, M.D., SEYMOUR Z. GOLDBLATT, M.D., STANLEY GOULD, M.D., JAMES L. HATLEBERG, M.D., HYUN-WHA KIM (Oh), M.D., SHEELAGH MARSH, M.D., WILLIAM R. O'REILLY, M.D., JOHN PAPATHANASIOU, M.D., FRANCISCO PEREZ-TURLAY, M.D., DONALD R. POHL, M.D., BYRON D. ROSEMAN, M.D., J. WILLIAM STOHLMAN, M.D., BELINDA C. STRAIGHT, M.D., LETICIA U. TINA, M.D., JOHN TKACZ, M.D., GHOLAM H. VAZINE, M.D., PIO G. VERA CRUZ, M.D., SIDNEY L. WERKMAN, M.D., LUCAS YAMMAMOTO, M.D., AND LILLIAN KOCH ZIEGLER, M.D.

PUBLICATIONS COMMITTEE OF THE MEDICAL STAFF: FREDERIC G. BURKE, M.D., PRESTON A. MCLENDON, M.D., E. CLARENCE RICE, M.D., SYDNEY ROSS, M.D., J. WILLIAM OBERMAN, M.D., AND GEORGE WILLIAM WARE, M.D.

PUBLISHED MONTHLY BY THE STAFF AND RESEARCH FOUNDATION OF THE CHILDREN'S HOSPITAL, WASHINGTON, D. C.

Cases are selected from the weekly conferences held each Friday at 12:30 P.M., from the Clinicopathological conferences and from the monthly Staff meeting.

This bulletin is printed for the benefit of the present and former members of the Attending and Resident Staffs, and the clinical clerks of Georgetown and George Washington Universities.

Subscription rate is $3.00 per year. Those interested make checks payable to "Clinical Proceedings Dept.," The Children's Hospital, Washington, D.C. Please notify on change of address.

Copyright 1958, Children's Hospital

Entered as second class matter November 21, 1946 at the post office at Washington, D.C., under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 538, Act of February 28, 1925, authorized January 17, 1947.

A REVIEW OF INCIDENCE, COMPLICATIONS, AND MANAGEMENT OF ECZEMA IN INFANTS AND CHILDREN

Hyun-Wha Kim (Oh), M.D.*

The word "eczema" is derived from the Greek and means "to boil out". "Eczema" is well known even to the layman, probably because of its chronicity, tendency to recur and refractoriness to treatment; indeed, this condition represents a major problem to the trio most interested: pediatrician, mother and infant.

Clinically, eczema is characterized by erythema, edema and vesiculation, followed by weeping and crusting. The term "eczema", as generally used, includes a wide variety of unrelated inflammatory dermatoses; in adults, contact dermatitis is commonly regarded as eczema, whereas in children, eczema is considered an atopic manifestation to some specific allergen usually thought to be an ingestant. This "atopic eczema" to which there is a certain amount of hereditary predisposition often precedes asthma or hay fever, and accounts for 75 per cent of all "eczemas" in childhood.

Atopic dermatitis was first described by Besnier in 1892 and is sometimes known as Besnier's prurigo. "Atopy" is Coca's term for human hypersensitivity. Other forms of "eczema", such as seborrheic dermatitis, infectious eczematoid dermatitis, nummular eczema, contact eczema, and rarely, eczematoid fungous infections, may also be seen in childhood.

OCCURRENCE OF ECZEMA IN INFANTS AND CHILDREN

The incidence of eczema in childhood varies according to the observer. Hill [1] has reported that about 75 per cent of all eczema in childhood is atopic dermatitis, and that about half of the infants who have atopic dermatitis later develop respiratory allergy. Epstein's [2] series of 100 cases of infantile eczema revealed the following incidence:

Atopic dermatitis	78
Seborrheic dermatitis	10
Contact dermatitis	6
Intertrigo	4
Dermatitis	2

In this series 58 were male, 42 were female. All patients were under two years of age. The earliest age of onset was one month, the latest, two years.

* Assistant Chief Resident.

Bain's [3] series of 168 cases of eczema occurring in infancy and childhood observed over a five year period revealed that 123 were infants under two years of age, while 45 were between the ages of two and 14 years. The ratio of males to females was 4:3. Two-thirds of these patients had a positive family history of some allergy. In Bain's cases, 25 per cent developed the skin lesions before one month of age, 64 per cent before three months of age, and 90 per cent before one year of age. Milk was the most common single offender, and its elimination offered the greatest chance for cure. Of the 124 children in this series who were under two years of age, breast milk was the only food being given to 22.4 per cent of these infants, at the time when the eczema developed; 15 per cent were on evaporated milk alone; and 69.2 per cent were on milk, orange juice, and cod liver oil. Bain emphasized the importance of an elimination diet in alleviating this condition.

Vowles, et al[4] presented a series of 84 patients with infantile eczema who had been followed by visits to the home for a period ranging from 13 to 32 years after initial hospital admission for infantile eczema. Six died in infancy from complicating infection. Of the remaining 78 only one-quarter had cleared completely by three years of age, but 55 per cent still had some evidence of the disease by 13 years of age; of this latter group a considerable proportion had persistence of their lesions by twenty years of age. Asthma, recurrent bronchitis or seasonal rhinorrhea were present in 73 per cent of this series as compared with 5 to 7 per cent of control groups. Fifty per cent had asthma alone as compared with 2 per cent of control groups. One or more attacks of pneumonia occurred in 22 per cent of patients but in only 2 per cent of control groups. Vowles stated that the tendency for eczema to persist is not influenced by birth weight, sex, color, sibling position, or type of feeding.

Birt[5] investigated 198 cases of infantile eczema observed since 1930. He observed that 25 per cent of infants had eczema up to or after the second year of life. Ratner[6] has stated that 50 to 60 per cent of his allergic patients had a history of onset of allergy in childhood. All infants with infantile eczema in his series demonstrated food sensitivity, but 59 per cent of the same infants reacted with other environmental substances. His figure of the average age of onset of eczema was 0.6 years whereas the average age of onset of asthma was three years. Osborne and Murrey[7] investigated 500 cases of atopic dermatitis and found 30 to 40 per cent of cases to be under one year of age. Seventy-five per cent underwent a summer remission; in two-thirds of these, wool was the dominant allergen. They stated their belief that wool may serve as a mechanical provocation as well as a contact and inhalant allergen and emphasized that atopic children should avoid contact with wool, corduroy, soap, and laundry starch.

Livingood and Shapiro[8] believe that contact dermatitis probably accounts for 15 to 20 per cent of dermatitis seen in children. Leider[9] uses the term "atopic habitus" to describe 3 to 10 per cent of the population who also have a tendency to an eosinophilia of 5 to 10 per cent. Epstein's[2] cases had an average eosinophilia of 9.7 per cent with a range of 1 to 29 per cent. Urbach and Jacobson[10] have stated that the majority of cases of infantile eczema (atopic dermatitis) end with spontaneous relief at about the age of two years or before. They believe that in a small percentage, the manifestations of the disease continue throughout childhood into adult life and that atopic dermatitis originating after two years of age is more likely to persist into adult life, to be chronic, and to be frequently accompanied by asthma or hay fever.

COMPLICATIONS OF ECZEMA IN CHILDHOOD

Since the advent of antibiotic therapy the incidence of complications of eczema in childhood has markedly decreased. However, instances of malnutrition, septicemia, otitis media, glomerulonephritis, pyelitis, pneumonia, multiple skin abscesses, and chronic skin infection with cutaneous pathogens, such as the Staphylococcus and Beta hemolytic streptococcus have been reported. The most feared complication is superimposed infection by the virus of either herpes simplex or vaccinia.

1. Eczema herpeticum (Kaposi's varicelliform eruption): This condition is due to infection with the herpes simplex virus superimposed on the eczematoid lesions. The condition is often fatal; encephalitis may be associated. Landtman, et al.[11] reported a ward epidemic affecting eight infants aged 6 weeks to 11 months. There was one death. Lesions occurred in skin areas previously affected with eczema and consisted of umbilicated vesicles surrounded by a narrow zone of erythema. Complement fixation testing revealed serologic evidence of infection with herpes simplex virus in four of six cases tested. Barton and Brunsting[12] reported two cases of eczema herpeticum occurring in adults; both recovered. Herzberg and and Pliess[13] reported a fatal case in a 37 year old man who contracted the condition from his wife who had herpes labialis. Three cases occurring in infancy were collected by Reyman [14]; all recovered.

2. Eczema vaccinatum: This condition is due to the lesions of cowpox superimposed on the eczematoid lesions and resembles generalized vaccinia. Death may occur. Vaccination against small pox is therefore avoided in an eczematous child and he is kept away from recently vaccinated persons, until all evidence of eczema has disappeared. Petersilge and Toomey[15] reported a fatal case of eczema vaccinatum in an eight month old white male infant with eczema who was exposed accidentally and indirectly to the pustule of an older sister who had been vaccinated. Dewar and Finn[16] reported that eczema vaccinatum developed in 2 of 84 pa-

tients who had "vaccination in the presence of skin disease". Both patients recovered. Ellis[17] reported two cases of eczema vaccinatum in three year old twin boys, both of whom had eczema. Both were exposed to a severe primary reaction to vaccination in an older brother. One twin died of eczema vaccinatum at the height of the primary reaction in the older brother; the other twin, despite a vaccination which evoked no response, developed eczema vaccinatum and recovered.

3. Hypoproteinemia and edema: Nisenson[18] in a series of 24 cases of severe eczema reported 20 patients with evidence of hypoproteinemia and eight with clinical evidence of edema. Factors predisposing to edema included extensive weeping of skin lesions, a diet poor in animal protein, and secondary infection. Albumin levels were low in 18 per cent, and globulin levels low in 5 per cent. A much lower level of the total serum proteins was necessary to produce edema in the child under one year than in the child over one year. In this series of 18 children under three years of age with severe eczema, six were under six months of age, five were aged six months to one year, and seven were aged one to three years.

4. Cataract and keratoconus: Brusting, Reed, and Bair[19] examined by slit lamp 1158 selected patients with atopic dermatitis and found typical cataracts in 136. In 79 of these the cataracts were either grossly visible or the patients were aware of visual impairment. Cataracts were unilateral in 39 instances. Ages of patients observed ranged from ten to 43 years. Atopic dermatitis antedated the cataracts in all cases; two-thirds of the patients had a strong family history of allergy.

Cataracts and keratoconus are among the less common but more striking complications of atopic dermatitis. The total incidence varies from three to ten per cent with sex distribution being roughly equal. Visual impairment occurs in about one-half of cases showing well defined cataracts. The typical atopic cataract begins in the late teens or early twenties and tends to progress during acute flareups of the underlying disease. Keratoconus is rare but occurs in young adults, is bilateral and is accompanied in one-half of the cases by cataracts.

Ingram[20] reported an incidence of juvenile cataracts of 16 per cent in 60 patients with atopic eczema, compared to the incidence of 15 to 20 per cent reported by others. In his series males predominated 3:2, two-thirds of the patients had a family history of allergy and 80 per cent had the condition from infancy.

5. Dermatopathic lymphadenitis: Larken[21] examined enlarged cervical lymph nodes removed from six children with infantile eczema and reported pathologic changes comparable to those described by Hurwitt[22] as dermatopathic lymphadenitis. The pathology of this condition shows some resemblance to that of Hodgkin's disease but is benign in nature

TABLE I

Complications of Infantile Eczema (after Epstein[2])

Complications on admission

Impetigo (non-specific)	6	Upper respiratory infection	4
Staphylococcic impetigo	3	Coryza	1
Streptococcic impetigo	2	Cold	1
Pyoderma	4	Intestinal infection	1
Erysipelas	1	Diarrhea	2
Furunculosis	1	Otitis media	2
Scabies	2	Asthma	3
Kaposi's varicelliform eruption	3	Rickets	1
Urticaria	1	Acrodynia	1

Complications occurring during hospitalization

Gastrointestinal upset	7	Diarrhea	1
Cold	5	Upper respiratory infection	1
Bronchitis	5	Pyoderma	1
Acute tonsillitis	1	Erysipelas	1
Otitis media	1		

and is thought to be a reactive response to the irritant skin disease. This type of adenopathy is usually refractory to treatment and chronic in course; marked leukocytosis (leukemoid reaction) and eosinophilia are usually associated.

Other complications: Of six deaths reported by Vowles, et al.[4] four children died at seven to eight months of age, one at 14 months of age, and one at 29 months. Cause of death was attributed to pneumonia in two, gastroenteritis in two, bronchitis and gastroenteritis in one, and undiagnosed disease in one. Schwartz[23] recorded ten deaths; four died of septicemia, four of fulminating pneumonia, and two of intestinal intoxication. All infants appeared quite toxic during their last illness. This toxemia was usually associated with rapid weight loss and dehydration. In a few, sudden collapse was the only prelude to death. Organisms implicated in the four infants with septicemia were Streptococcus in two, Streptococcus and Pneumococcus in one, and Pneumococcus alone in one.

Epstein[2] reported the complications in 100 children hospitalized with infantile eczema. Complications on admission to the hospital and contracted during the hospital stay are recorded in Table I. Complications contracted at the hospital were nearly all infections of the respiratory or gastrointestinal tract and the incidence of complications occurring in the hospital was 23 per cent.

No deaths occurred in Epstein's series; he emphasized that death oc-

curring in a patient with eczema is avoidable. He noted that the incidence of dangerous complications such as severe respiratory infection and erysipelas had been markedly reduced since the advent of the sulfonamides.

Davies[24] reported three patients with infantile eczema under six months of age who died suddenly while in the hospital. No adequate cause of death was found. Other observers think that the causes of death are usually fulminating and overwhelming infection.

MANAGEMENT OF ATOPIC DERMATITIS IN INFANTS AND CHILDREN

A) Specific measures (Urbach and Jacobson[10]):

1) Detection of allergens by means of detailed history, elimination procedures and skin tests. Allergens include ingestants (foods and drugs), inhalants (pollens, dusts, danders), and contactants (pollens, dusts, chemicals, bacteria).

2) Elimination of specific allergens: Between the ages of six months and two years foods are the most common demonstrable allergens. Elimination diets are therefore important in therapy but must be constructed so that good nutrition is maintained. Cow's milk, egg, wheat, citrus fruits, tomatoes, fish, and fish oils are the foods most commonly incriminated. In eczema occurring after the age of two years, environmental allergens such as house dust, wool, feathers, animal dander, cottonseed, and silk are occasionally found to excite or aggravate symptoms. Practically speaking, it is sometimes difficult to eliminate these specific allergens (e.g., pollen during the pollen season).

3) Hyposensitization: Urbach and Jacobson believe that the classical allergic approach to the management of atopic dermatitis by hyposensitization techniques is, for the most part, unsuccessful except for the rare case of well established hypersensitivity to pollen, dust or other inhalants. In the case of foods hyposensitization is disappointing; elimination diets are far more successful.

B) Non-specific measures:

1) Topical management: Urbach and Jacobson[10] believe that the management of eczema in childhood may be successfully handled by the empirical use of topical preparations but emphasize that potent preparations should be used cautiously to minimize aggravation of the disease. They recommend the following topical preparations according to the stage of the lesion.

a) Wet Dressings:

Zephiran® chloride 1:2000 (Benzalkonium)	one teaspoonful of Zephiran concentrate in one quart of water.

Burow's solution	one tablespoonful to one glassful of water.
Potassium permanganate	two grain tablet; one tablet in one gallon of water (1:25,000).

b) Lotions:
Calamine lotion (U.S.P.)

c) Pastes:
1-2-3 paste:

Burow's solution	10.0
Aquaphor®	20.0
Lassar's paste	30.0

d) Emollients:
Hydrogenated vegetable oils (Crisco®, Spry®)
Lubriderm® (Texas Pharmacal)
Nivea oil® (Duke Pharmacal)
Acid Mantle cream® (Dome)
Plastibase® (Squibb)

e) Active Medicinals:
Vioform® ointment—3 per cent (Ciba)
Vioform® cream—3 per cent (Ciba)
Neosporin® ointment (Burroughs-Wellcome)
Icthammol—3 per cent in zinc oxide ointment.
Pediatric Daxalan® (Dome): active ingredient 1.4 per cent crude coal tar.
Kolpix A® (Dome): active ingredient: 2 per cent coal tar.
Kolpix D® (Dome): active ingredient: 2 per cent coal tar.
Hydrocortisone ointment—1-2 per cent (Cort-Dome®, Cortef®, etc.)

f) Paints:
Zetar® (Dermik): active ingredient colloidal crude coal tar.

g) Baths:
Almay Tar Bath® (Almay Corp.): active ingredient oil of cade.

h) Soap substitutes:
Phisoderm® (Winthrop Stearns)
Phisohex® (Winthrop Stearns)

2) Antibiotics: The systemic and occasionally the topical use of antibiotics is important in controlling secondary infections of atopic eczema. Bactericidal and bacteriostatic topical preparations are frequently used to control secondary infections in eczematoid areas but those agents which are potent sensitizers, or which are frequently used systemically should not be used topically (penicillin, streptomycin, sulfonamides). In addition, extensive areas of secondary infection should never be treated topically but the appropriate systemic route used instead (Urbach and

Jacobson). Bacitracin ointment and Vioform® are recommended by many for topical use.

3) Sedation: Sedatives are frequently necessary both to enable the infant to sleep and to obviate damage to the skin produced by orgiastic scratching episodes.

4) Psychiatric therapy: Hill[1] has doubted that deep-seated emotional disturbances have much to do with the causation of atopic dermatitis in infants but agrees with other authorities that emotional tension may aggravate eczema in older children. He believes psychotherapy may be indicated for those cases with long-standing psychiatric difficulties.

5) Hospitalization: Hospitalization offers both advantages and disadvantages to the child with eczema. In general, hospitalization is indicated when the eczema is so severe, or extensive, or complicated by infection that proper care cannot be given at home, or when the mother is exhausted.

6) Nutrition: A nutritious, well-balanced diet should be offered at all times. As many observers have noted, food sensitivity is the most common etiologic factor in atopic dermatitis of children under two years of age, and elimination diet is necessary when the specific allergen is identified. However, the elimination diet should not be practiced to an extreme, and complete compensation in nutrition should be carried out. Finkelstein[25] has felt that the presence of animal protein increased the itching, or as he called it, the angioneurotic pruriginous component of eczema. He recommended that the diet should consist primarily of fruit, vegetables, and vegetable protein. However, there are many who do not agree with him. Nisenson[18] recently emphasized the importance of feeding meat, animal protein, at an early stage of eczema, particularly where soy milk is used exclusively. The use of soy increased the incidence of protein deficiency when used as milk substitute because of the fact that cysteine is a necessary amino acid in protein synthesis and soy is low in cysteine.

Nisenson's series reviewed eight cases of edema associated with atopic dermatitis. Total protein in the serum ranged from 1.7 gm. per 100 ml. to 5.0 gm. per 100 ml. In these eight cases edema was reversible, usually improving within one to three weeks on a diet high in meat protein, Amigen®, or Nutramigen®.

Hill[1] has stated that although removal of certain foods from the diet sometimes helps the baby with eczema, this practice should not be carried to an extreme. He has emphasized that the child must have an adequate well-balanced diet no matter whether it causes eczema or not. The more rapidly a baby gains weight the more active is his eczema likely to be. Usually the eczematous baby is fat, and so he should be prevented from getting any fatter; contrawise, if a baby with eczema is too thin he should

be made fatter. Hill stated that many cases of eczema in babies are due more to eating too much in general than to any specific food sensitivity.

7) Antipruritics: Antipruritics are a very necessary part of therapy; the itching of atopic eczema is intolerable and constant scratching not only prevents healing but also increases the hazard of secondary infection. Topical preparations containing phenol, menthol, camphor, coal tar, or benzocaine may be used. Antihistaminic drugs such as Benadryl® or Pyribenzamine® may be used locally or systemically; these drugs frequently have an additional sedative action.

8) Restraint and bandaging: The main purpose of restraint and bandaging is to put the skin at rest and to give it a chance to heal. A large amount of the skin trouble in any baby or child with eczema is caused by what he does to himself. No skin can heal when it is continually scratched and rubbed. The bandages should be considered as part of the child's clothes and put on daily. Whatever salve is being used is first applied, then a soft piece of white cotton cloth (never gauze), then 2 inch elastic bandage. When the bandages are taken off it is best to take off only one at a time, apply the salve and put the bandage on again before taking off another one so that the baby cannot scratch.

In many hospitals the baby is "spread-eagled", that is, his wrists and ankles are wound with sheet wadding and then tied by tapes to the sides of the crib. However, these babies struggle against their bands very vigorously and it is not entirely possible to prevent a baby from scratching and rubbing no matter what is done; many mothers will not use this technique on their babies.

Elbow splints may be used to prevent the baby from scratching his face. Sometimes when the baby's arms and legs are bandaged and the rest of the body is covered by clothes he cannot do much effective scratching. Little cotton cloth bags are sometimes put over the hands or the hands are put into the toe of a small white cotton stocking and then the other end of the stocking is pinned to the shirt so that even if the baby does get his hands into contact with his skin he cannot dig it with his fingernails. Of course, fingernails of any eczematous baby or child should be cut short.

However, it is not possible to prevent rubbing of the face on the shoulders. Eczema of the popliteal and antecubital spaces of older children should always be bandaged to prevent trauma.

9) Bathing: It is sometimes said that babies with eczema should never be washed with water but if water does not irritate the baby or his skin, there is no harm in using it. However, eczematous skin must be handled very gently without irritation. If there are breaks in the skin and the baby is likely to cry when he is put into water, sponge baths of the diaper

area, axillae and groins can be given. If eczema of the arms and the legs are being bandaged it is best to keep these areas dry.

Starch baths can be given to the baby with eczema who is not sensitive to starch.

10) Soap: Soap is, in general discouraged for use on eczematous skin, especially active tender areas. Hypoallergenic soap such as Lowila® is recommended by many authorities. Hill[1] stated that he had never seen eczema in a baby or child caused by mild soap nor had he seen any harm done to eczematous children by such mild soap except when it is used on an oozing erythematous area. However, if soap does irritate the skin he recommended Phisoderm® or Phisohex® if any infection is present.

11) Nursing care and hygiene: These must be fastidious and accompanied by both attention and affection during hospitalization, as well as in the home.

12) Vaccinations: Hill[(1)]

a) No vaccination against smallpox should be given to the eczematous child or to any of his siblings.

b) The eczematous child should not be allowed to come into contact with anyone infected with herpes simplex virus; if the mother is suffering from "fever blisters", mother and child must be separated.

c) No influenza vaccine should be given to the eczematous child who is known to be sensitive to egg white.

13) Ultraviolet light: Ultraviolet light may be tried in chronically thickened and dry skin. Ultraviolet light may occasionally help older children who have "winter skin" (thick and dry), and who are nearly free from eczema in the summer. These children are usually at their worst in the spring. Hill uses ultraviolet light when he is at his wit's end and does not know what else to do, or when he thinks the parents need treatment. He states that ultraviolet light treatment makes the parents feel that they are really doing "something" and ultraviolet light actually helps the child, particularly his itching. He feels the best discussion of light treatment is by Nexmand[(26)] who treated 57 patients (mostly children) with atopic dermatitis at Finsen Institute in Copenhagen. The light baths were given every other day. If the patient tolerated the treatment 30 to 40 baths were given, depending on the condition of the skin. Of 57 patients treated 33 were considerably improved.

14) Adrenocorticosteroids: Steroid therapy when properly used has a place in the management of infantile eczema. Steroids are of limited value, however, since the eczema "rebounds" after their withdrawal; such "rebound" eczema is, moreover, harder to control. Hill[(1)] treated 12 children with eczema with local hydrocortisone ointment (one to two and one-half per cent). He felt that hydrocortisone ointment does not cure the

eczema but does reduce inflammation to a marked degree. This is especially true for superficially inflamed, slightly scaly areas. Lesions reappeared after discontinuation of the ointment. Hill also treated about 60 eczematous children with oral cortisone, using a maximum dose of 75 mg. a day to infants and young children and gradually reducing the dosage. He concluded that steroids are useful in the treatment of infantile eczema but should not be considered a cure.

For infants under two years of age 0.3 gm. of potassium chloride was prescribed daily; for older children added salt in the diet was prohibited. Hill's belief is that the use of large doses of cortisone for such a disease as eczema is not justified since the complications arising from large doses are frequently more severe than the original disease.

Nexmand[26] reported two cases of chronic eczema in adults treated with cortisone and ACTH. This therapy brought about temporary relief but upon the withdrawal of medication a diffuse exudative papular dermatosis appeared which was greater in intensity than the original eczema. Sternberg and Newcomer[27] treated 24 cases of severe atopic eczema with a daily dose of 200–300 mg. of cortisone administered either orally or intramuscularly for a period of ten to 14 days. Complete healing was produced in all but one patient who could not tolerate the medication; this patient subsequently responded to corticotropin therapy. They reported that intramuscular cortisone was most effective. Oral cortisone was also satisfactory but a somewhat higher dose and more frequent administration was required. All patients relapsed after systemic cortisone therapy was discontinued. The authors concluded that systemic cortisone therapy should be reserved for severe cases of eczema in which all conventional modalities of treatment have failed to arrest the progress of the disease. In these cases the authors stated that cortisone therapy should be limited to four to six weeks. In mild to moderate cases of atopic dermatitis they felt that systemic cortisone therapy is contra-indicated due to the rebound skin disorder.

Debre, et al.[28] have reported results of 46 infants and children with eczema treated with systemic cortisone and aspirin. Initially, cortisone was used followed by maintenance therapy with cortisone and aspirin, and finally cortisone alone. These authors report 42 per cent of patients completely cured at the end of initial treatment, 18 per cent markedly improved, and 30 per cent partially improved.

Hill[1] has suggested the use of cortisone in eczema as follows:

a) In the oozing crusted rash which is kept inflamed by rubbing to such an extent that the usual methods of treatment are of no value.

b) In the three to five year old child with a diffuse deep-seated generalized papular eruption, to give a "breathing spell" while locating the etiologic factors, if possible.

c) In the older child with eczema in whom the eczema is so severe that nothing else can be done with it. In this type of case both mother and child are so exhausted that a potentially serious family situation frequently arises.

15) Synthetic vitamin A: Stoesser and Nelson[29] treated ten children with eczema aged six months to ten years with synthetic vitamin A in a daily dose of 25,000 to 200,000 units for a period of three to 21 months. Nine tolerated the medication, and of these, six were decidedly improved and three partially improved. There were no signs of toxicity to the vitamin. These authors concluded that synthetic vitamin A (palmitate) is valuable in treating infantile eczema in children who are allergic to or cannot tolerate fish.

SUMMARY AND CONCLUSIONS

Death resulting from one of the complications of infantile eczema is not rare. It is emphasized that special attention should be paid to preventing or minimizing these complications of eczema in childhood by means of proper nursing care, gentle topical treatment, judicious use of antibiotics and avoidance of contact with the viruses of herpes simplex or vaccinia.

REFERENCES

1. Hill, L. W.: The Treatment of Eczema in Infants and Children. C. V. Mosby Company, St. Louis, 1956.
2. Epstein, S.: Hospital Morbidity and Mortality of Infantile Eczema. J. Pediat., **26:** 541, 1945.
3. Bain, K. P.: Eczema in Infancy and Childhood. South. M. J., **34:** 863, 1941.
4. Vowles, M., et al.: Infantile Eczema; Observation on Natural History and Prognosis. Brit. J. Dermat., **67:** 53, 1955.
5. Birt, A. R.: The Value of Elimination Diets in the Treatment of Infantile Eczema. Canad. M. A. J., **43:** 520, 1940.
6. Ratner, B.: Allergy in Childhood. III. Its Onset and Natural Progress. J. A. M. A., **111:** 2345, 1938.
7. Osborne, E. D., and Murrey, P. F.: Atopic Dermatitis; a Study of Its Natural Course and of Wool as a Dominant Factor. Arch. Dermat. & Syph., **68:** 619, 1953.
8. Livingood, C. S., and Shapiro, E. M.: Dermatitis in Infants and Children; Diagnosis and Management. Pediatric Dermatology, Pediatric Clinics of North America. August, 1956.
9. Leider, M.: Practical Pediatric Dermatology. C. V. Mosby Company, St. Louis, 1956.
10. Urbach, F., and Jacobson, C.: Atopic Dermatitis. Pediatric Dermatology, Pediatric Clinics of North America. August, 1956.
11. Landtman, B., et al.: Kaposi's Varicelliform Eruption; Report of a Ward Epidemic. Ann. Paediat., Fennae, **1:** 61, 1954–1955.
12. Barton, R. L., and Brunsting, L. A.: Kaposi's Varicelliform Eruption. Arch. Dermat. & Syph., **50:** 99, 1944.
13. Herzberg, J. J., and Pliess, G.: Eczema Vaccinatum Mit Tödlichen Verlauf. Frankfurter Zeitschrift Für Pathologie (Munchen), **66:** 330, 1955.

14. Reyman, F.: Pustulosis Varicelliformis Kaposi. Ugeskrift For Laege, **118:** April–June 1956.
15. Petersilge, C. P., and Toomey, J. A.: Death Caused by Vaccinia in an Eczematoid Infant. Ann. Paediat., **61:** 454, 1944.
16. Dewar, W. A., and Fin, O. A.: Vaccination in the Presence of Skin Diseases. Glasgow M. J., **35:** 141, 1954.
17. Ellis, F. A.: Eczema Vaccinatum; Its Relation to Generalized Vaccinia. J. A. M. A., **104:** 1891, 1935.
18. Nisenson, A.: Hypoproteinemia and Edema in Eczema. J. Pediat., **46:** 544, 1955.
19. Brunsting, L. A., Reed, W. B., and Bair, H. L.: Occurrence of Cataracts and Keratoconus with Atopic Dermatitis. A. M. A. Arch. Dermat., **72:** 237, Sept. 1955.
20. Ingram, J. T.: Besnier's Prurigo. Brit. J. Dermat., **67:** 43, 1955.
21. Larken, V. D., et al.: Dermatopathic Lymphadenitis in Infantile Eczema. J. Pediat., **24:** 442, 1944.
22. Hurwitt, E.: Dermatopathic Lymphadenitis: Focal Granulomatous Lymphadenitis Associated with Chronic Generalized Skin Disorders. J. Invest. Dermat., **5:** 197, 1942.
23. Schwartz, A. B.: The Cause of Death in Infantile Eczema. J. Pediat., **4:** 174, 1934.
24. Davies, J. H. T.: Sudden Death in Infantile Eczema; an Indispensable Factor in Its Production. Brit. J. Dermat., **2:** 182, 1940.
25. Finkelstein, H.: Dietetic Treatment of Eczema in Infants and Children. Am. J. Dis. Child., **54:** 344, 1937.
26. Nexmand, P. H.: ACTH and Cortisone Withdrawal Causing Diffuse Papular Dermatoses. Danish M. Bull. (Copenhagen), July, 1955.
27. Sternberg, T. H., and Newcomer, V. D.: Treatment of Atopic Dermatitis with Cortisone. J. A. M. A., **148:** 904, 1952.
28. Debre, et al.: Treatment of Infantile Eczema. Arch. Franc. Pediat., **13:** 1, 1956.
29. Stoesser, A. V., and Nelson, L. S.: Synthetic Vitamin A in Treatment of Eczema in Children. Ann. Allergy, 703, Nov.–Dec., 1952.

RECTAL PROLAPSE OF AN ILEOCECAL INTUSSUSCEPTION

Byron D. Roseman, M.D.*

Intussusception is by no means uncommon. It occurs most frequently in the age period from six to 18 months, usually in spring and summer, in apparently healthy infants, and is rare in the neonatal period. More than half of the cases described are in boys.

Beyond this no general discussion of intussusception is warranted. Many excellent reviews of the topic are to be found,[1, 2, 3] including two from this institution[4, 5].

CASE REPORT

D. A. M., a four month old white girl entered Children's Hospital for the first time with a history of diarrhea of 24 hours duration and a rectal prolapse. The infant had always strained at stool and produced "small, hard, round balls" of feces. She also was said to have colic manifested by episodes of screaming without apparent cause,

* Assistant Chief Resident.

FIG. 1. Showing prolapsed intussusceptum with ileo-cecal valve evident

accompanied by a drawing up of the legs. From birth she had regurgitated varying but usually small amounts of her formula. In an attempt to correct these difficulties the formula had been changed first from evaporated milk and water to homogenized milk, then to soybean milk. For one week prior to admission the baby had seemed unwell, crying fairly continuously. During the month prior to admission she had gained only 6 ounces. On the day before admission she vomited once and that evening passed two large, liquid, brown stools. During the night she passed six more stools, some of which were blood-streaked, and contained mucus. On the day of admission she vomited once again, and passed six small stools which continued to be blood-streaked and mucinous. Her private physician administered penicillin intramuscularly, and kaolin with pectin by mouth on the morning of admission. Several hours before hospital admission, while being held, she was observed to be straining as if she were having a bowel movement. Subsequent to this an apparent rectal prolapse was discovered. This was reduced by her physician who then admitted her to the hospital.

The patient was a product of an uncomplicated gestation and normal delivery of a primiparous mother. Birth weight was 8 pounds, 15 ounces.

At admission she appeared to be a well developed, well nourished, attractive, chubby female infant who was not in obvious distress but appeared pale and somewhat lethargic. Her rectal temperature was 101.2 degrees, pulse 160 per minute and respirations 50 to 70 per minute. The abdomen was rotund, and was consistent with the infant's physique. It was soft to palpation and no muscle spasm or intra-abdominal organs or masses were evident. Peristaltic sounds were not abnormal. External genitalia were normal. Considerable bloody mucus was smeared over the buttocks. No rectal examination was done other than replacement of an ostensible rectal prolapse.

Admission urinalysis showed 10 mg. of albumin per 100 ml., a moderate amount of acetone, and 0 to 2 white blood cells per high power field. Blood hemoglobin was 13.0 gm. per 100 ml., hematocrit 40 per cent, white blood count 15,650 per cu. mm. with 27 per cent segmented neutrophiles, 65 per cent mature lymphocytes, 7 per cent eosino-

philes, and 1 per cent monocytes. Carbon dioxide combining power on admission was 13.5 mEq./L. On the day following admission, after intravenous fluids had been given, blood chloride was reported as 98.5 mEq./L. Stool culture subsequently was found to be devoid of pathogenic organisms. During the night of admission she vomited four times, the vomitus at first consisting of yellow-white material, then dark-brown fluid, then "coffee grounds". She passed one liquid green and two small, pink mucinous stools.

By the morning following admission the patient had received 420 ml. of fluid intravenously. She was examined and another "prolapse" was found and reduced. Investigation for intussusception was then begun. This clinical impression was given added weight by the finding of a left lower quadrant mass. X-ray of the abdomen at this time showed distended loops of small bowel, and ascending and transverse colon. There was no gas in the descending colon, sigmoid and rectum.

She was seen by a consultant in surgery who advised immediate operation for a probable ileo-colic intussusception with prolapse from the rectum. In the operating room the intussusception was again replaced; about eight inches of bowel had prolapsed from the rectum and the ileocecal valve was visible (Fig. 1). A balloon catheter was inserted into the rectum and the balloon inflated. When the abdomen was opened through a right rectus incision the intussusceptum was noted to have progressed through the entire ascending, transverse, and decending colon, curling up the colon as it progressed. The visible ileocolic intussusceptum was noted to be at the level of the upper sigmoid. This was mobilized and brought into the incision site, while an operative assistant elevated a water can attached to the balloon catheter to a height of 2 feet, initiating reduction of the intussusception. There was no evidence of tumor, enlarged mesenteric nodes, polyps, or diverticulum. There was no apparent bowel necrosis although the terminal ileum appeared edematous. The ascending colon was then fixed in the right parietal gutter with two interrupted silk sutures. During surgery the patient received 100 ml of whole blood. Postoperative course was uneventful. The infant was discharged, eating well, having normal stools, active and playful on the seventh hospital day.

DISCUSSION

Most of the literature offers little on the incidence of prolapsed intussusception. Occasionally cases are described or alluded to [1, 3, 6, 7, 8, 9, 10], so that it is apparently not rare. Ravitch and McCune[1] noted the occurrence of this complication of intussusception in 11 of 152 cases (7 per cent), and Ladd and Gross[8], in 4 cases of 484 (0.8 per cent).

The lesion is important in that it may be confused readily with rectal prolapse, particularly if encountered for the first time. Differentiation of procidentia from rectal prolapse of an intussusceptum has been emphasized by several authors[1, 6, 7, 8]. *Distinguishing the two is a simple matter. In intussusception the finger may be inserted between the protruding mass and the rectal sphincter. In rectal prolapse the digit cannot be passed between the mass and anal margin.*

It is a matter of urgency that the diagnosis be made. There is an ". . . increase in the probability of death as the leading point approaches the anus which probably depends on the increasing tension and damage to the

mesentery as well as the increasing difficulty in operative treatment of the lesion"[8].

SUMMARY AND CONCLUSIONS

A small proportion of cases of intussusception encountered in pediatric practice will present with prolapse of the intussusceptum through the anus. The case of a 4 month old white girl who recovered following such a catastrophe is described. For approximately 24 hours the lesion was thought to be a rectal prolapse but was subsequently diagnosed correctly as an intussusceptum. Procidentia is easily differentiated from prolapse of an intussusception by digital rectal examination. Mortality and morbidity are directly proportional to the extent to which the intussusceptum advances toward the rectum.

REFERENCES

1. Ravitch, M. M., and McCune, R. M., Jr.: Intussusception in infants and Children. J. Pediat., **37:** 153, 1950.
2. Lawrence, G. H., and Ulfelder, H.: Intussusception; a Review of Experience at the Massachusetts General Hospital, 1937–1951. New England J. Med., **247:** 499, 1952.
3. Cope, Z.: The Early Diagnosis of the Acute Abdomen. Oxford University Press, London. 10th Ed., pp. 139, 1951.
4. Ware, G. W., and Coffey, R. J.: Intussusception in Infancy and Childhood. Surg., Gynec., & Obst., **91:** 173, 1950.
5. Bregman, R. L.: Intussusception; Case Review. Clin. Proc. Child. Hosp., **11:** 83, 1955.
6. Mentzer, C. G.: Anorectal Disease. Pediat. Clin. North Am., pp. 113, February 1956.
7. Turell, R.: Progress in Pediatrics; Pediatric Proctology. Am. J. Dis. Child., **79:** 510, 1950.
8. Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood. W. B. Saunders, Philadelphia, 1941.
9. Fitzwilliams, D. C. L.: Pathology and Etiology of Intussusception from the Study of 1000 cases. Lancet, **1:** 628 (Feb. 29), 1908; Ibid.; Lancet, **1:** 709 (Mar. 7), 1908.
10. Duvanant, D., and Wilson, H.: Intussusception of the Appendix with Complete Inversion of the Appendix and Protrusion from the Anus. Am. Surg., **135:** 287, 1952.

VIRUSES AS A CAUSE OF GASTROENTERITIS

Viola ~~Mae Young~~, Ph.D.*

Shigella enteritis, *Salmonella* enteritis and the more recently discovered colienteritis[1] are well established entities, but the viral enteritides have only begun to receive attention from the scientific worker. The term "viral

* Gastroenteritis Studies, Bact. Dept., Walter Reed Army Institute of Research, Washington, D. C. Visiting Lecturer, Stritch School of Medicine, Chicago, Illinois.

enteritis" has been used for many years by medical men who suspected that viruses cause some of the gastroenteritis syndromes for which no other etiologic agent could be determined. Intensive investigation in this field is a relatively new development and has only become possible with the discovery of new techniques for virus studies.

Prior to the adaptation of tissue cultures to virus cultivation a number of investigators had studied outbreaks of non-specific gastroenteritis utilizing the older viral techniques. Lembcke, et al.[2] injected ether extracts of stool specimens from a diarrheal outbreak intraperitoneally into suckling Swiss mice and observed that a lethal agent was present. Light and Hodes[3] inoculated filtrates and suspensions of fecal samples intranasally into calves and observed a subsequent bloody diarrhea. Buddingh and Dodd[4] produced transmissible lesions on the rabbit cornea by the inoculation of filtrates of diarrheal stools. Cummings[6] later demonstrated that stool filtrates from normal infants could produce these results. Though Meiklejohn[5] and Cummings[6] confirmed the calf inoculation studies of Light and Hodes, the discovery by Baker[7] of a pneumonenteritis virus in calves, and later of 2 strains of virus causing diarrhea in cattle[8], has raised the question of interpretation of earlier findings.

With the development of tissue culture methods for virus cultivation and the application of these techniques to the study of fecal samples, however, a rapidly increasing number of enteric viruses came to light. These viruses were divided by Sabin[9] into two main categories: "1) those known to be the cause of specific diseases, e.g., poliomyelitis, herpangina (Coxsackie A), epidemic pleurodynia (Coxsackie B), the pharyngeal-conjunctival syndrome (APC), herpes simplex, and presumably also those of infectious hepatitis, mumps and influenza B; 2) the large number of recently recognized viruses whose relationship to various disease syndromes is still a matter of current and future investigations." Several of those viruses known to give rise to specific diseases have either been proven to cause, or are suspected of being able to cause symptoms of gastroenteritis, particularly in infants. For example, polio viruses undoubtedly initiate enteritis symptoms in infants and young children more frequently than central nervous system involvement.

Group A Coxsackie viruses are reported to have been associated with some cases of "virus diarrhea." Travassos[10] reported an outbreak in Rio de Janeiro among 25 infants, 22 of whom presented gastrointestinal disturbance and 4 of whom had neurological symptoms. Nine strains of Coxsackie Type A were isolated from the 20 fecal specimens examined and 4 strains were recovered from 21 throat washings. Pohjanpelto[11] isolated Coxsackie viruses, chiefly A-4, from 4.4 per cent of 187 patients with diarrhea and from only 1.1 per cent of the healthy controls. Both of these investigators ascribe significance to their findings in disagreement with

Parrott[12] who found only 2.4 per cent of 165 children with undiagnosed diarrhea had Group A viruses present in their stools and ascribed no etiological significance to them. As he pointed out, however, "That is not to say . . . that diarrhea may not occasionally be a symptom of herpangina, or of other Group A infections."

Between July and September, 1955, an outbreak of pharyngo-conjunctival fever, chiefly in school children, swept through Germany into Switzerland. Some of the patients complained of vomiting, diarrhea and abdominal pain as well as of nose, throat and conjunctival inflammation. Adenovirus Type 3 was isolated from many of these patients. Of the first 50 infants and children with gastroenteritis submitting samples from Children's Hospital to the Walter Reed Army Institute of Research for "gastroenteritis" studies 70 per cent had symptoms referable to the upper respiratory system. Similar observations have been reported elsewhere. McCorkle, et al.[13] noted the high incidence of gastrointestinal symptoms occurring at the same time as common respiratory disease in a group of Cleveland families. No firm conclusion regarding the relation of virus to diarrhea can be based on this type of evidence, and the investigation of this question must be pursued by more exacting methods.

The agent which causes infectious hepatitis is considered a true intestinal tract inhabitant. Though its cultivation has recently been reported by Rightsel, et al.[14] these findings await further confirmation. Careful epidemiologic studies have shown that with infection by this particular virus diarrheal episodes occur perhaps more frequently in children than hepatic involvement. Some observers consider infectious hepatitis to be chiefly a disease of children. The lack of a suitable experimental host has left much unsettled concerning this disease and what role, if any, it plays in the general picture of infant diarrhea.

A similar sort of problem exists with the infectious non-bacterial gastroenteritis agents studied by Gordon and co-workers[15-19]. Gordon has been unable to propagate these agents in trials involving a number of types of tissue culture, animals or embryonated eggs. Serial passages have been carried out in human volunteers, however, with reproducible symptoms and the development of immunity against reinfection. Two different infectious non-bacterial gastroenteritis agents apparently exist. The febrile strain (FS) has an average incubation period of 27 hours, presents severe constitutional symptoms, but seldom causes diarrhea. The afebrile (Marcy) strain has an average incubation period of 50 hours and, though it produces less severe constitutional symptoms, a severe watery diarrhea typically occurs. These points of difference, together with the fact that there is no cross immunity, provide proof that two agents are involved. These are the only non-bacterial agents known thus far which can be considered proved etiological agents of the syndrome of gastroenteritis in humans.

The second category of enteric viruses mentioned by Sabin[9] includes the "large number of recently recognized viruses whose relationship to various disease syndromes is still a matter of current and future investigations." These viruses began to attract attention when tissue culture techniques were first applied to large scale studies of fecal samples from cases of paralytic poliomyelitis and later to non-paralytic polio. When a number of agents cytopathogenic for monkey kidney tissue cultures were discovered which proved to be unrelated to any known virus, a committee was formed to study the problem of their classification. This committee named the new viruses "enteric cytopathogenic human orphan" (ECHO) viruses[19], the term "orphan" referring to the fact that they are not related to any known disease. There are now 19 such viruses accepted by the Committee on the ECHO Viruses and undoubtedly many more have been isolated but are still incompletely studied. Results of data collected by a number of different investigators indicate that several types of ECHO viruses, notably ECHO type 6[20-22] and ECHO type 9[24-26] are apparently the etiologic agent in some cases of aseptic meningitis. A collection of data further supporting the etiologic potential of ECHO type 9 was recently presented by McLean, et al.[27]. Meyer[20] found that a high percentage of his aseptic meningitis cases from whom ECHO type 6 virus was isolated had gastrointestinal symptoms such as nausea, vomiting, abdominal pain and mild diarrhea during the first few days of illness. It does not seem unreasonable to assume that such gastrointestinal symptoms might occur in the absence of meningeal symptoms.

In a study completed in 1956, Ramos-Alvarez[28] of Cincinnati discovered a high incidence of cytopathogenic agents (43 percent) from rectal swabs of 56 children with diarrhea as compared to a recovery rate of only 6 per cent among children without diarrhea who had visited clinics and hospitals for other reasons. Twenty-nine per cent of the agents recovered were known ECHO viruses, 17 per cent were Coxsackie viruses, 12.5 per cent were untyped Coxsackie viruses, another 12.5 per cent were polio type I viruses and 25 per cent were new antigenically distinct ECHO viruses. One adenovirus type 3 was also isolated.

In a similar study carried out by the Gastroenteritis Studies Group of the Bacteriology Department of the Walter Reed Army Institute of Research, 48 per cent of 29 Puerto Rican infants with gastroenteritis yielded agents cytopathogenic for monkey kidney tissue culture. The patients varied from 16 days to 3¼ years of age; 21 of 29 specimens were collected from infants under six months of age. Neutralization tests revealed that five of the patients had a significant rise in antibody titer to the virus they were harboring.

Though identifications are incomplete, at least half of the viral agents are known ECHO viruses, one is a member of the Coxsackie group, two are

apparently new viruses and one is related to the D1 agent of Ramos-Alvarez. No polio viruses were isolated from the group of specimens.

The high percentage of ECHO viruses recovered from children with diarrhea as compared to those without diarrhea[28] would appear to indicate that they are associated with the disease. That such viruses are not the viral counterpart of normal intestinal bacterial flora is suggested from the decreasing rate of recovery with increase in age. Sabin[9] reports that in surveys carried out in Cincinnati 5.2 per cent ECHO viruses were recovered from the one to four year group, 2.6 per cent from the five to nine age group, only 0.2 per cent in the 10 to 14 age group and none at all in the 15 to 17 year olds. These viruses are only rarely isolated from stool specimens from normal adults. Continued research is essential to the proper evaluation of the pathogenic potential of the many new agents being isolated.

Some of the problems of the laboratory diagnosis of viral diarrhea may be summarized as follows: 1) How much viral diarrhea is caused by known agents such as polio, Coxsackie, adenoviruses and perhaps infectious hepatitis? 2) What is the etiological significance of the recently isolated viruses associated with diarrhea? 3) How can we recover and identify viral agents which, according to clinical observations, we must presume to exist? 4) What is the possible overall effect of the viral agents on known bacterial pathogens?, and 5) What might be the influence of viral episodes on immunity and reaction to subsequent contact by the same virus?

REFERENCES

1. Young, V. M.: Colienteritis from "Diseases of Children in the Subtropics and Tropics." Edward Arnold (Publishers) Ltd., London. To be published.
2. Lembcke, P. A., Quinlivan, J. J., and Orchard, N. G.: Epidemic Diarrhea of Newborn: Report of Two Outbreaks. Am. J. Pub. Health, **33:** 1263, 1943.
3. Light, J. S., and Hodes, H. L.: Studies on Epidemic Diarrhea of the Newborn; Isolation of a Filtrable Agent Causing Diarrhea in Calves. Am. J. Pub. Health, **33:** 1451, 1943.
4. Buddingh, G. J., and Dodd, K.: Stomatitis and Diarrhea of Infants Caused by a Hitherto Unrecognized Virus. J. Pediat., **25:** 105, 1944.
5. Meiklejohn, G.: Viral Studies on Etiology of Epidemic Diarrhea of the Newborn. California Med., **67:** 238, 1947.
6. Cummings, G. D.: Epidemic Diarrhea of Newborn from Point of View of Epidemiologist and Bacteriologist. J. Pediat., **30:** 706, 1947.
7. Baker, J. A.: A Filtrable Virus from Pneumonia and Diarrhea of Calves. Cornell Vet., **32:** 202, 1942.
8. Baker, J. A., York, C. J., Gillespie, J. H., and Mitchell, G. B.: Virus Diarrhea in Cattle. Am. J. Vet. Research, **15:** 525, 1954.
9. Sabin, A. B.: The Significance of Viruses Recovered from the Intestinal Tract of Healthy Infants and Children. Ann. New York Acad. Sc., **66:** 226, 1956.
10. Travassos, J., DePaola, D., Echanis, J. S., Bruno-Lobo, M., Vasconcellos, J. V., and DeGoes, P.: Occurrence of Coxsackie Virus in Rio de Janeiro. I.

Preliminary Studies on the Isolation of the Virus From Feces and Throat Washings. Anais Microbiol., **2:** 71, 1953.

11. Pohjanpelto, P.: The Coxsackie Group of Viruses, Epidemiological Studies. Ann. Med. Exper. et Biol. Finniae, **33:** (Suppl. 6), 1, 1955.
12. Parrott, R. H.: The Clinical Importance of Group A Coxsackie Viruses. Ann. New York Acad. Sc., **67:** 230, 1957.
13. McCorkle, L. P., Badger, G. F., Curtiss, L., Dingle, J. H., Hodges, R. G., and Jordan, W. S., Jr.: A Study of Illness in a Group of Cleveland Families. XII. The Association of Respiratory and Gastrointestinal Symptoms; an Estimation of the Magnitude and Time Relations of the Association. Am. J. Hyg., **64:** 357, 1956.
14. Rightsel, W. A., Keltsch, R. A., Tekushan, F. M., and McLean, I. W., Jr.: Tissue-culture Cultivation of Cytopathogenic Agents from Patients with Clinical Hepatitis. Science, **124:** 226, 1956.
15. Gordon, I., Ingraham, H. S., and Korns, R. F.: Transmission of Epidemic Gastroenteritis to Human Volunteers by Oral Administration of Fecal Filtrates. J. Exper. Med., **86:** 409, 1947.
16. Jordan, W. S., Jr., Gordon, I., and Dorrance, W. R.: A Study of Illness in a Group of Cleveland Families. VII. Transmission of Acute Non-bacterial Gastroenteritis to Volunteers; Evidence for Two Etiologic Agents. J. Exper. Med., **98:** 461, 1953.
17. Gordon, I., Meneely, J. K., Currie, G. O., and Chicoine, A.: Clinical Laboratory Studies in Experimentally Induced Epidemic Non-bacterial Gastroenteritis. J. Lab. & Clin. Med., **41:** 133, 1953.
18. Gordon, I., Patterson, P. R., and Whitney, E.: Immunity in Volunteers Recovered from Non-bacterial Gastroenteritis. J. Clin. Investigation, **35:** 200, 1956.
19. Committee on the ECHO Viruses, National Foundation for Infantile Paralysis. Enteric Cytopathogenic Human Orphan (ECHO) Viruses. Science, **122, 3161:** 1187, 1955.
20. Meyer, H., Jr., Rogers, N., Miesse, M., and Crawford, I.: Aseptic Meningitis Caused by Orphan Viruses and Other Agents. Ann. New York Acad. Sc., **67:** 332, 1957.
21. Karzon, D. T.: Discussion: Part III ECHO Viruses. Ann. New York Acad. Sc., **67:** 351, 1957.
22. Svedmyr, A.: Discussion: Part III ECHO Viruses. Ann. New York Acad. Sc., **67:** 352, 1957.
23. Kibrick, S., Meléndez, L., and Enders, J. F.: Clinical Associations of Enteric Viruses with Particular Reference to Agents Exhibiting Properties of the ECHO Group. Ann. New York Acad. Sc., **67:** 311, 1957.
24. Rotem, C. E.: Meningitis of Virus Origin. Lancet, **1:** 502, 1957.
25. Garnett, D. G., Burlingham, A., and Van Zwanenberg, D.: An Outbreak of Aseptic Meningitis of Virus Origin in East Suffolk. Lancet, **1:** 500, 1957.
26. Boissard, G. P. B., Stokes, L. J., McCrae, A. D., and MacCallum, F. O.: Isolation of Viruses Related to ECHO 9 from Outbreaks of Aseptic Meningitis. Lancet, **1:** 500, 1957.
27. McLean, D. M., and Melnick, J. L.: Association of Mouse Pathogenic Strain of ECHO Virus Type 9 with Aseptic Meningitis. Proc. Soc. Exper. Biol. & Med., **94:** 656, 1957.
28. Ramos-Alvarez, M.: Cytopathogenic Enteric Viruses Associated with Undifferentiated Diarrheal Syndromes in Early Childhood. Ann. New York Acad. Sc., **67:** 326, 1957.

infant feeding in the hospital

your selection is easy...

milk-formula feedings

Lactum®
"Instant" Powder/Liquid
the classic milk and carbohydrate formula in ready-prepared form

protein-generous feedings

Olac®
"Instant" Powder/Liquid
ready-prepared formula with generous protein

flexible feedings

Dextri-Maltose®
Powder
the professional carbohydrate modifier

milk-sensitive infants

Sobee®
"Instant" Powder/Liquid
hypoallergenic soya formula

protein-sensitive infants

Nutramigen®
Powder
ready-prepared formula containing protein in hydrolyzed form

in digestive disorders

Probana®
Powder
ready-prepared formula for use in non-specific digestive disorders

The Mead Johnson Formula Products Family offers you a formulation and form for each infant feeding need

for house and discharge formulas

• carbohydrate modifier • liquid formulas • powdered formulas • all with simple basic dilutions • easy to mix, easy to autoclave

To help save you time in nursery, formula room and obstetric department, Mead Johnson printed services include Nursery Record Book, Formula Room Handbook, instructions for mothers and other materials. Ask your Mead Johnson Representative, or write to us, Evansville 21, Indiana.

Mead Johnson
Symbol of service in medicine

FPS77

MEAD JOHNSON & COMPANY, Suite 422 Eig Building, 8641 Colesville Road
Silver Spring, Maryland. JUniper 9-1222